done under local anesthesia, especially after abdominal sections, and apparently result from emboli; (5) we have to consider the possibility of the aspiration of infectious material into the alveoli of the lungs.

Operative pneumonia has been reported with the following frequency:

	Operation.	Pneumonia.	Mortality.
Anders	57,842	46	
Hewitt	2910	3	
Rumboll	1500	6	
Gurlt	52,177	30	
Derjuskinsky	4946	43	0.4%
Kuemmell	1017	40	28
Henle	1787	143	65
Bibergeil	3909	135	
Schultze	5724	27	
Kelly	1800	8	
Rothrock	489	5	3
Silk	5000	13	
Gebele	1196	77	53
Total	140,397	570	(0.4%) (Mortality 45%)

Anyone, I think, who has the courage to try the fresh-air treatment, and sufficient persuasive power over the patient's relatives to allow it to be tried, will, I am convinced, never abandon it, even though the future may bring us a specific for pneumonia in the form of a curative serum. Of course, it takes time to eradicate prejudices and to change beliefs. It has not been so very long since patients with tuberculosis were advised to spend their time in closed rooms before a stove. It is an even shorter time since pneumonia patients were swathed in poultices, enveloped in jackets, and kept in rooms in which even the windows and doors were made as air-exclusive as possible by stuffing cotton into the cracks. We have gradually dispelled the fear of cold sponging and bathing in typhoid fever, and we will in time abate the dread of fresh air in pneumonia.

I shall never forget a big negro, in the third day of his pneumonia, whom I saw in the general medical ward, restless, fevered, and panting, the typical picture of the anguish which one so often sees in these cases. An hour later I saw him in the pneumonia ward whither he had been promptly transferred. It was a cold day in March, the windows and the door were wide open, so that there was a constant current of fresh cold air blowing over the beds. The patient greeted me with a smile, displaying a fine line of teeth which contrasted strikingly with his Ethiopian blackness, and remarked, "Fo de Lawd, Doc, I feels lak anoder man down hyar." This is not an isolated or exceptional incident, numbers of patients desperately ill have told me, upon inquiry, that they felt "pretty good." If then we can show not only an increased *bien faisançe* on the part of the patient, but also a reduced mortality, and an absence of deleterious effects why should not this method of treatment be the usual instead of the exceptional one?

THE PHARMACOLOGY OF HEART STIMULANTS.[1]

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A SUBJECT so broad as that covered by the title of this paper cannot be considered within a reasonable limit of time, and I shall therefore confine my remarks to those drugs which are classed together as the digitalis group, and to those pharmacological points which are of interest especially to the medical therapeutist. In discussing this subject I shall consider first one or two noteworthy facts common to the whole group, and, then, the points of individual difference of its more important members.

There are three essential factors in the action of these drugs which interest clinicians: (1) The stimulant influence upon the cardio-inhibitory mechanism which prolongs the diastole and thus slows the pulse; (2) the increase in the tonicity of the heart muscle leading to a more complete and more powerful systole; and (3) the constriction of the bloodvessels. The dicrotism, the incomplete diastole, and the incöordination of the heart, which are produced by very large doses of digitalis, are toxic manifestations and of minor importance to the therapeutist.

The action of digitalis in slowing the pulse is, I believe, a much more important factor in its beneficial influence in heart disease than is generally deemed. It is evident that if the quantity of blood thrown out by the heart at each contraction were constant, then the more slowly the heart beat the less blood would be pumped in a given time; in other words, if the volume of the pulse remained the same the rate of the pulse would be an accurate criterion of the work performed, and an inhibitory stimulation would mean less work done by the heart and consequent reduction in the blood pressure. But the volume of blood expelled at each contraction does not remain the same. The longer the diastole, within reasonable limits, the larger is the volume of the pulse wave. It has been shown experimentally by Elving and Wendt[2] and others that there is a degree of compensatory correlation between the rate and the volume of the pulse so that the amount of blood pumped by the heart per minute tends to remain constant despite variations in its rate.

The mechanism of this arrangement is not difficult to apprehend if we remember that the slowing of the pulse depends chiefly upon the prolongation of diastole. The lengthening of the period of auricular relaxation allows more blood to flow from the great veins into the auricle, either of the right or left heart as the case may be; and the prolongation of the ventricular diastole permits the auricle completely to empty its contents into the ventricle, so that when that chamber begins to contract it finds itself well filled with blood, and consequently the pulse volume is increased. Although according to the experiments quoted the increased volume is ordinarily not sufficient entirely to compensate for the diminution in the pulse rate which results from stimulation of cardiac inhibition and the amount of blood per minute pumped may be somewhat lessened, nevertheless, it seems to me that the heart beating at a slow rate is working more economically than the rapidly acting organ.

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[1] Read at the meeting of the American Therapeutic Society, Philadelphia, May 7, 1908.

[2] Skand. Arch. f. Physiol., 1907, xix, p. 96.

If the ventricle begins to contract at a time when it is only half filled with blood, half of the effort of contracting is wasted in reducing the size of the chamber to correspond to the small amount of blood which is within it. On the other hand, if the ventricle is completely filled with blood before it begins to contract it is evident that all of the shortening of the cardiac muscle fiber which takes place does useful work; therefore the bigger the pulse volume the more work does the heart accomplish with the same expense to its total power, and consequently the more slowly the heart acts the more sparing is it of its reserve energy, and it obtains as well a longer period for rest and reconstruction.

In cases of chronic heart disease, when it is essential to make the burden thrown upon the feeble heart as light as possible, it appears to me highly important that we do what we can to slow the pulse, and the inhibitory stimulation of digitalis becomes *the* beneficial factor in its effects. The mere stimulation of the heart muscle is a matter of minor importance. What necessity is there for, or advantage derived from, the elevation of blood pressure? Dyspnœa, local congestion, or œdema may be temporarily relieved by other heart stimulants, as caffeine or ammonia; but is the patient any farther from the grave because some local evidence of heart weakness is concealed?

The objection has been raised against this view, that inhibitory stimulation tends to a relaxation of the muscle which might encourage a further dilatation of a weak cardiac wall, but here the action of digitalis in increasing the tonicity of the heart muscle comes into prominence as a preventive of this danger.

The only one phase of the effect of digitalis in increasing the muscular vigor of the heart, which I think needs emphasis at this time, is that in no other portion of the heart is the influence of the drug felt in the same way as it is in the left ventricle. Although the observation of Kaufmann that the disatolic, as well as the systolic, pressure in the left ventricle is augmented by digitalis indicates that the force of the auricular systole is to an extent increased by digitalis, yet it is a common occurrence to see the ventricle of the frog's heart firmly contracted in systolic spasm, while the dilated engorged auricles are making ineffectual efforts to force blood into the larger cavity.

This fact becomes of importance in certain cases of mitral regurgitation when the ventricular muscle has undergone a larger degree of hypertrophy than has the auricle. Under the influence of digitalis the ventricle may drive the blood backward through the leaky mitral valve so vigorously as to overcome the muscular resistance of the feeble auricle and lead to further dilatation of that part of the heart. Moreover, in these cases the second defense, the right ventricle, will likely prove insufficient, for the experiments of Bayet[3] as well as my own studies[4] have shown that the action of the drug upon the right heart is comparatively insignificant.

There remains of the physiological actions of digitalis to be considered, its vasoconstricting influence. This action has frequently been regarded as a harmful by-effect to be avoided or combated, a point of view which I believe has at times led practitioners to adopt irrational or even injurious measures.

It is of course true that it requires more force to drive the blood through narrow arteries than it does through dilated ones, but it is equally true that in many cases the vascular contraction is a conservative effort on the part of nature to maintain the circulatory equilibrium. The coronary arteries are comparatively small vessels springing at right angles from a large trunk, a condition which is in many ways unfavorable for the entrance of blood into them. It is evident, therefore, that a considerable degree of pressure in the aorta is an absolute requisite for good coronary circulation. Tigerstedt and Johansson[5] have shown that in states of very low arterial tension despite the diminished resistance in front of it the ventricle may be unable to empty itself as completely as normally, a phenomenon most rationally attributed to lessened heart power the result of insufficient nutrition.

I do not mean to infer that the vascular effects of these remedies is never detrimental. There is scarcely room for doubt that when, as a result of degenerative changes in the muscle or an extreme degree of dilatation, the muscular tissue of the ventricle is too scanty to respond properly to the tonic influence of digitalis, the increased resistance to the flow of blood may work great damage. But I do think that these cases are much less frequent than is generally believed. Clinicians should not be too hasty in their efforts to dilate the vessels in cases of chronic heart disease; especially so since almost the only agents which we possess for this purpose, the nitrites, are substances which increase the rate of the pulse and must therefore tend to counteract the beneficial influence of digitalis in slowing the heart. The only reason that more harm has not been accomplished by the of administration, and partly by the love of the novel, clinicians have from time to time experimented on their patients with various of the glucosides which have been found in the digitalis leaves. At present digitoxin holds the foreground among these substances; but this much is clear, that if there be any single principle which represents completely the therapeutic virtues of digitalis that principle is *not* digitoxin. In the first place Fraenkel has shown that of all the digitalis glucosides digitoxin is the most dangerous and the most cumulative in its action; secondly, the work of Gottlieb and Magnus would indicate that digitoxin is decidedly more active in its vasomotor effects than either digitalis itself or strophanthus; and, thirdly, Kakowski experimenting with digitalin, digitalein, and digitoxin, was able with none of them to produce the increase in the contracting power of the heart that is brought about by digitalis.

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[3] Acad. Royale de Med. Belg., 1892.

[4] Amer. Jour. Physiology, 1902, vi, p. 283.

[5] Skand. Arch. f. Physiol., 1891, ii, p. 409.

ATAXIA OF THE HEART MUSCLE.

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Since the simultaneous graphic registration of venous and arterial pulses has been generally adopted, following the pioneer work of Mackenzie,[1] we have learned that dissociations in the activity of the different heart chambers occur very much more frequently than we formerly supposed. The dissociation of auricular and ventricular contractions has been generally accepted since the publications of His, Mackenzie, and many other writers. The anatomical cause has been shown to consist in a lesion of the bundle of His, reproduced experimentally by Hering[2] and Erlanger[3] and shown pathologically by Stengel,[4] and Ophuls and myself.[5]

Only lately the discussion of the possibility of hemisystole, an isolated contraction of one side of the heart, while the other remained in diastole, has been taben up.

Twenty years ago von Leyden[6] reported several cases in which he tried to explain by a hemisystole the phenomena observed in cases of mitral disease. In those cases two apex beats and two pulsations of the jugular vein corresponded to one arterial pulse; the first apex beat with a corresponding arterial and venous wave was supposed to correspond to a complete contraction of both heart chambers; while the second apex beat with a venous wave, but without an

[1] Study of the Pulse.

[2] Pflüger's Archiv., 1905, cviii.

[3] Jour. of Exper. Med., 1906.

[4] Amer. Jour. Med. Sci., 1905.

[5] Deut. Arch. f. klin. Med., vol. lxxxvii.

[6] Ztschr. f. klin. Med., vol. lxiv.

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